	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001032928	A2	20010510	WO 2000-US30474	20001103
	WO 2001032928	A3	20020725		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI	US 1999-165398P	P	19991105
	US 2000-196571P	P	20000411

AB The invention discloses methods, gene databases, gene arrays, protein arrays, and devices that may be used to det. the hypersensitivity of individuals to a given agent, such as drug or other chem., in order to prevent toxic side effects. In one embodiment, methods of identifying hypersensitivity in a subject by obtaining a gene expression profile of multiple genes assocd. with hypersensitivity of the subject suspected to be hypersensitive, and identifying in the gene expression profile of the subject a pattern of gene expression of the genes assocd. with hypersensitivity are disclosed. The gene expression profile of the subject may be compared with the gene expression profile of a normal individual and a hypersensitive individual. The gene expression profile of the subject that is obtained may comprise a profile of levels of mRNA or cDNA. The gene expression profile may be obtained by using an array of nucleic acid probes for the plurality of genes assocd. with hypersensitivity. The expression of the genes predetd. to be assocd. with hypersensitivity is directly related to prevention or repair of toxic damage at the tissue, organ or system level. Gene databases arrays and app. useful for identifying hypersensitivity in a subject are also disclosed.

=> d his

(FILE 'HOME' ENTERED AT 21:16:24 ON 30 NOV 2003)

FILE 'USPATFULL' ENTERED AT 21:16:41 ON 30 NOV 2003

L1	1596	S	(GLUCOCORTICOSTEROID OR GLUCOSTEROID OR CORTICOSTEROID OR STE
L2	5	S	L1 (P) MICROARRAY#
L3	22089	S	(GLUCOCORTICOSTEROID OR GLUCOSTEROID OR CORTICOSTEROID OR STE
L4	41591	S	(GLUCOCORTICOSTEROID# OR GLUCOSTEROID# OR CORTICOSTEROID# OR
L5	47	S	L4 (5A) RESPONSIVENESS
L6	10	S	L5 (P) (RNA OR MRNA OR NUCLEIC OR CDNA OR DNA OR OLIGONUCLEOT
L7	3	S	L5 (9A) DETERMINING

FILE 'MEDLINE, BIOSIS, CAPLUS' ENTERED AT 21:23:04 ON 30 NOV 2003

L8	554259	S	(GLUCOCORTICOSTEROID# OR GLUCOSTEROID# OR CORTICOSTEROID# OR
L9	1884	S	L8 (5A) RESPONSIVENESS
L10	4	S	L9 (9A) DETERMINING
L11	35929	S	L8 (9A) RESPONS?
L12	374	S	L11 (9A) DETERMIN?
L13	226		DUP REM L12 (148 DUPLICATES REMOVED)
L14	16	S	L13 AND (RNA OR MRNA)
L15	202	S	L13 AND PY<2001
L16	202		DUP REM L15 (0 DUPLICATES REMOVED)
L17	20	S	L11 AND SAA#

ab

L18 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN

Full Text | Citing References

AN 2002:506123 CAPLUS
DN 137:123900
TI Differential glucocorticoid enhancement of the cytokine-driven transcriptional activation of the human acute phase serum amyloid A genes, **SAA1** and **SAA2**
AU Thorn, Caroline F.; Whitehead, Alexander S.
CS Department of Pharmacology and Center for Pharmacogenetics, University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA
SO Journal of Immunology (2002), 169(1), 399-406
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The human acute phase serum amyloid A (A-**SAA**) genes, **SAA1** and **SAA2**, have a high degree of sequence identity that extends ~450 bp upstream of their transcription start sites. Each promoter contains analogously positioned functional binding sites for the transcription factors NF-κB and NF-IL6. In human HepG2 hepatoma cells transfected with **SAA** promoter luciferase reporter constructs, administration of IL-1 and IL-6, singly or in combination, induced **SAA1** and **SAA2** transcriptional readouts that were qual. indistinguishable. However, under induced conditions, the **SAA2** promoter had a significant quant. transcriptional advantage over the **SAA1** promoter. The application of the synthetic glucocorticoid dexamethasone in the context of cytokine stimulation enhanced the transcriptional activity of the **SAA1**, but not the **SAA2**, promoter such that readout from the former became equiv. to that from the latter. A putative glucocorticoid response element (GRE) is present (between residues -208 and -194) only in the **SAA1** gene; a similar sequence in the corresponding region of the **SAA2** gene is disrupted by a nine-residue insertion. The **SAA1** GRE was shown to be functionally active and the **SAA2** disrupted GRE was shown to be functionally inactive in expts. using reporter constructs carrying **SAA1** and **SAA2** promoters that had been modified by site-specific mutagenesis. Quant. anal. of transcript-specific RT-PCR products, derived from **SAA1** and **SAA2** mRNAs after treatment of HepG2 cells with cytokines in the presence or absence of dexamethasone, confirmed that the endogenous **SAA1** gene has a cytokine-driven transcriptional disadvantage that is superseded by a marginal transcriptional advantage when glucocorticoids are present.
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2003 ACS on STN

Full Text | Citing References

AN 2001:338762 CAPLUS
DN 134:362292
TI Methods of determining individual hypersensitivity to a pharmaceutical agent from gene expression profile
IN Farr, Spencer
PA Phase-1 Molecular Toxicology, USA
SO PCT Int. Appl., 222 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

L2 ANSWER 1 OF 1 MEDLINE on STN
AN 96185283 MEDLINE
DN 96185283 PubMed ID: 8606597
TI Presymptomatic diagnosis of familial steroid-resistant nephrotic syndrome.
AU **Fuchshuber A**; Janssen F; Gribouval O; Niaudet P; Kamoun A; Antignac C
SO **LANCET**, (1996 Apr 13) 347 (9007) 1050-1.
Journal code: 2985213R. ISSN: 0140-6736.
CY ENGLAND: United Kingdom
DT Letter
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199605
ED Entered STN: 19960531
Last Updated on STN: 19960531
Entered Medline: 19960517

L8 ANSWER 20 OF 47 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1996:378928 BIOSIS
DN PREV199699101284
TI The glucocorticoid insensitivity syndrome.
AU Lamberts, S. W. J.
CS Dep. Med., University Hospital Dijkzigt, Dr. Molewaterplein 40, NL-3015 GD Rotterdam, Netherlands
SO Hormone Research (Basel), (1996) Vol. 45, No. SUPPL. 1, pp. 2-4.
CODEN: HRMRA3. ISSN: 0301-0163.
DT Article
LA English
ED Entered STN: 26 Aug 1996
Last Updated on STN: 26 Aug 1996
AB Recent studies demonstrate that primary (hereditary) abnormalities in the glucocorticoid receptor gene make 6.6% of the normal population relatively 'hypersensitive' to glucocorticoids, while 2.3% are relatively 'resistant'. These abnormalities might explain the well-known phenomenon that some individuals develop severe adverse effects during therapy with a low dose of glucocorticosteroids, while others do not develop side effects even during long-term therapy with a much higher dose. This heterogeneity in glucocorticoid sensitivity in the normal population might eventually allow the prediction of a 'safe' dose of glucocorticosteroids in individual patients. 'Resistance' to the beneficial clinical effects of glucocorticosteroid therapy in some patients with severe rheumatoid **arthritis** and asthma is probably seldom related to generalized primary (hereditary) glucocorticoid resistance. In most patients this 'resistance' seems to be acquired and localized to the inflammation sites, where it is caused by high local cytokine production which interferes with glucocorticoid action. Recognition of localized, acquired glucocorticoid resistance is of great importance, as alternative drug therapy with other immune-modulating drugs, such as cyclosporin and methotrexate, should be considered. Chronic high-dose glucocorticosteroid treatment in such patients insufficiently reduces symptomatology, while generalized side effects occur, as the rest of the body of the patient has a normal sensitivity to these drugs.